

stage II, 27.9% (n=6 295) at stage III, and 39.0% (n=8 820) at stage IV. There were no information regarding stage for 14.0% (n=3 171) of cases. Mortality/ incidence ratio has improved from 0.88 in 1980 to 0.76 in 2007. First year's lethality increased from 55.1% in 1980 to 67.2% in 2007. Five year observed survival of patients with gastric cancer decreased from 22.3% for patients diagnosed in 1980 to 14.3% for patients diagnosed in 2004. The age-standardized incidence rates (European standard) decreased from 36.9 per 100 000 population in 1980 to 22.1 in 2007, but mortality – from 31.8 in 1980 to 16.5 in 2007. The same trend was observed if analyzed by gender.

Conclusions: Incidence of gastric cancer and its caused mortality has a trend to decrease in Latvia. However it is still considerably higher than on average in European Region or in European Union. The proportion of late diagnosed cases is high and indicates on necessity to improve early diagnostics and to perform more effective actions against cancer.

P-0025 P53-ARG72PRO POLYMORPHISM CONFERS ANY SUSCEPTIBILITY TO GASTRIC CANCER IN ARDABIL

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Background: Gastric Cancer (GC) as the third most common malignancy in Iran, accounts for ~50% of all GI cancers who cause 55% of all cancer-related deaths in Iran. The rates of GC reported from Ardabil Province are among the highest in the world. Upper gastrointestinal cancer accounts for more than 50% of all cancer deaths in this area. Codon 72 polymorphism of the tumor suppressor gene TP53 has been associated with a higher risk in the development of several types of cancer. The polymorphism results in a variant protein with either an arginine (CGC) or a proline residue (CCC). We aimed analyze the association of the TP53 codon 72 polymorphism with the risk of developing gastric cancer in a high-risk population around the world.

Methods: We enrolled 100 patients with mean age 65.9 Yrs. affected with primary GC and same age- and sex- matched healthy control participants. The analysis has been carried out by PCR-RFLP on DNA extractions from peripheral blood.

Results: In the case group, the genotype was 16.1%, 42.5%, and 41.4% for Arg/Arg, Arg/Pro, and Pro/Pro, respectively. And for controls were 18.5%, 40.2%, and 41.4%. In comparing case and control group, any significant difference has been found.

Conclusion: Because of high frequency of GC in our province, the investigations about the role of genetic susceptibilities for GC are very important. In spite of finding no relationship between P53 polymorphisms, studying other genetic variations is recommended.

P-0026 CLINICAL CHARACTERISTICS OF GASTRIC CANCER PATIENTS WITH A FAMILY HISTORY

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Background: A family history of gastric cancer is considered to be a possible etiologic factor. This study was designed to analyze the clinicopathological characteristics of gastric cancer patients with a family history of gastric cancer and to aid in planning diagnostic and therapeutic approaches in such patients

Method: From January 1997 to July 2001, 611 gastric cancer patients who underwent surgery at our center were enrolled in this study. We divided two groups of patients with a family history of cancer and patients without a family history of cancer. We compared age, location of lesion, number of lesion, grossly and microscopically histological type, TNM stage, extent of gastrectomy and CEA level between two groups.

Results: The highest range of group with a family history of gastric cancer were more younger than that of the patients without a family history of cancer. (p=0.05) The solitary lesion of gastric cancer was not statistically significant difference in two groups. But, multiple primary lesions (more than 2) was more common with family history (p=0.05). The positive family history group had higher incidence compared to negative gastric cancer family history in EGC IIc type. (16.7% vs 6.9%)(p=0.032). There were no statistically significant difference in the location between the two groups. In the evaluation by histologic distribution, 'signet ring cell type' showed the highest distribution in the group with family history (19 cases, 31.7%) while 'poorly differentiated type' showed the highest distribution in the group without family cancer (214 cases, 39.3%); however, the difference showed no statistical significance (p=0.11). There were no statistically significant difference in the TNM stage and extent of gastrectomy in the two groups. 9 patients (14.5%) showed CEA elevation of more than 5 ng/ml in the group with family history compared to 138 patients (25.1%) in the group without family history. 53 patients (85.5%) showed CEA elevation of less than 5 ng/ml in the group with family history compared to 411 patients (74.9%) in the group without family history. There was no clinically significant difference between two groups. 5-years survival rate in the group with family history was 55.0% compared to 60.5% in the group without family history. There was no statistically significant difference between two groups.

Conclusions: There was no statistically significant difference in sex, age distribution, location of cancer lesions, extent of gastrectomy, CEA level and 5-year survival rate

depend-ing on the presence of family history of gastric cancer in patients. Only gross differentiation showed statistically significant difference, and multiple primary lesions (more than 2) was more common in the group with family history.

P-0027 SERUM PEPSINOGEN LEVELS IN DEVELOPING GASTRIC CANCER SCREENING APPROACHES FOR IRANIAN HIGH RISK POPULATIONS

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Background: Besides dramatic decrease of gastric cancer (GC) incidence in developed countries during recent years, it has been repeatedly reported as the second most prevalent cancer with the highest mortality rate in developing countries like Iran. Several studies have shown that serum pepsinogen (PG) levels can be used to screen subjects at high risk of GC development. The aim of this study was to evaluate the role of serum pepsinogen I and II levels as two potential markers for non-invasive screening of high risk populations.

Materials and Methods: 382 GC patients (cases), 626 non ulcer dyspeptic patients (NUD) and 179 healthy blood donors were enrolled as hospital and population based controls, respectively. Fasting blood samples were taken for measuring serum PG I, PG II by commercially available ELISA tests according to the manufacturer's instructions. GC cases were categorized according to tumor subsite and subtype. PMN/neutrophil infiltrations, gastric atrophic and intestinal metaplastic changes were graded according to the newly developed OLGA staging system.

Results: Serum PG I, II levels, I/II ratio were significantly different among cases and controls (P<0.05). Multiple logistic regression analyses showed that low PG I/PG II ratio (≤ 3.0) increases the risk of GC development by 5.2-7.7, which is mostly owed to cardia than non-cardia GC when compared to population based controls (OR=4.7; 95%CI=2.0 -10.7 vs. OR=3.6; 95%CI=1.5-8.5) and hospital based controls (OR=4.3; 95%CI=2.2-8.4 vs. OR=3.3; 95%CI=1.7-6.7). When cases were stratified according to GC subtypes, low PG I/II ratio also presented a risk for GC development, which was more pronounced for intestinal than diffuse tumor subtypes, when healthy (OR=5.6; 95%CI=2.43-12.8 vs. OR=2.9; 95%CI=1.1-7.5) and NUD (OR=5.15; 95%CI=2.7-10.0 vs. OR=2.7; 95%CI=1.9-6.04) controls were selected as the reference groups. Microscopic grading of inflammation in gastric biopsies among NUD group demonstrated that PGI/II ratio is significantly (P<0.001) lower in those who suffer from severe grades of gastric inflammation (grade 3: 10.6 \pm 15.4; grade 4: 19 \pm 31.7) in comparison with those with lower grades of inflammation (grade 0: 15.1 \pm 15.2; grade 1: 10.5 \pm 5.15; grade 2: 11.8 \pm 11.43).

Conclusion: The statistically significant risk impact of low serum PGI/II ratio on GC development in both subsites and subtypes, recommends the application of this non-invasive assay in population screening approaches and identification of high risk subjects for follow-up endoscopy and early detection of malignant lesions.

P-0028 THE IMPACT OF JOINT POSSESSION OF SINGLE NUCLEOTIDE GENE POLYMORPHISMS ON SUSCEPTIBILITY TO GC DEVELOPMENT IN IRAN

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Introduction: Gastric cancer is a multi-factorial disease resulting from interaction between host, pathogen and environmental factors. It has been speculated that geographic variations in gastric cancer risk may be partly due to single nucleotide gene polymorphisms in genes causing susceptibility to cancer. The aim of this study was to assess the impact of a number of gene polymorphisms and gastric cancer risk in an Iranian population.

Materials and Methods: We evaluated the joint effect of several single nucleotide polymorphisms (SNPs) on gastric cancer development and its subcategories. Ten gene polymorphisms involved in inflammation (interleukins: IL-1 β , IL-1RN, IL-8, IL-10), detoxification of carcinogens (glutathione s-transferases M1, T1: GSTM1, GSTT1), folate metabolism (methylene tetrahydrofolate reductase: MTHFR), and intercellular adhesion (E-cadherin: CDH-1) were evaluated by by CTPP-PCR and PCR-RFLP on genomic DNA extracted from white blood cells. In this study, 98 patients diagnosed with gastric cancer (GC) were compared with 140 healthy individuals using logistic regression analysis, calculating odds ratios (ORs) and 95% confidence intervals (95% CI) by SPSS 18.0 statistics software.

Results: We observed that individuals with 5 or more SNPs were at more than two fold increased risk of gastric cancer (OR: 2.255, 95% CI: 1.006-5.055, P=0.048) which was further enhanced in the older (>50 years) age stratum (OR: 3.632, 95% CI (1.324-9.965), P=0.012). In addition, stratification based on GC subsites demonstrated that